concentration (MIC) >10 mg/l), ciprofloxacin (MIC 16 mg/l) and tetracycline (MIC 64 mg/l) but sensitive to spectinomycin (MIC 32 mg/l) and cefuroxime (MIC 0·32 mg/l). The strain carried plasmids of 3·0MDa and 25·2MDa and the latter was shown to carry the Dutch type *tet*M tetracycline resistance determinant by polymerase chain reaction. Typing studies revealed that the strain belonged to the prototrophic auxotype and the IB7 serovar. The chlamydia antigen assay was negative.

At the end of the course of ofloxacin the patient re-attended the clinic with a persisting urethral discharge. A Gram-stained smear of the discharge revealed intracellular Gram-negative diplococci. Culture of a urethral swab yielded an organism indistinguishable in all respects to the previous isolate. On this occasion the patient was treated with a 2 g *stat* im dose of spectinomycin. On follow up seven days later only a slight discharge was present and both smear and culture were negative for gonococci although polymorphonuclear lymphocytes were seen in the former. He was therefore treated as a case of post-gonococcal urethritis and given a 10 day course of 250 mg oxytetracycline four times daily.

When first introduced ciprofloxacin had exceptional in-vitro activity against strains of *N gonorrhoeae*[1-3] and consequently has been increasingly used as a first line treatment for gonorrhoea. However, a treatment failure with another quinolone, enoxacin, was reported some time ago.[4] In the UK, strains with decreased sensitivity (MIC $\geqslant$ 0·05 mg/l) to ciprofloxacin have been detected since 1989 and treatment failures with ciprofloxacin have been associated with some of these infections.[5,6] More recently reports from the Philippines and Thailand[7,8] have revealed strains with ciprofloxacin MIC of >1 mg/l and in the case of the Philippines at least 10% of strains had a ciprofloxacin MIC $\geqslant$ 0·25 mg/l. Sentinel studies in the USA have revealed the importation of gonococci with ciprofloxacin MIC of 2 mg/l into Hawaii from SE Asia and also revealed 14% of strains in Ohio to have MICs between 0·13 mg/l and 0·25 mg/l.[9] In 1994 Birley *et al*[10] reported the failure of a 5 day course of twice daily 250 mg doses of ciprofloxacin in a case of gonorrhoea caught in Spain. This infection was also caused by a strain with high-level resistance to ciprofloxacin (MIC 16 mg/l).

Since 1988 216 strains of gonococci with an MIC of ciprofloxacin $\geqslant$ 0·05 mg/l have been referred to the PHLS Gonococcus Reference Unit and 13 have had an MIC >1 mg/l; none of the strains with reduced sensitivity also had high-level resistance to tetracycline, but 61% were penicillinase producers. Local incidence of ciprofloxacin resistance remains low, in 1994 only one of 338 county of Avon isolates was resistant (MIC 1 mg/l), this was not a penicillinase-producer and the infection was contracted in the UK.

This case re-emphasises the importance of culture for cases of gonorrhoea in order to be able to test the antibiotic sensitivities of the organism and thus assist the selection of suitable chemotherapeutic agents. The need for continued vigilance for ciprofloxacin resistance is reinforced, especially where the patient was infected in the Far East.

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Epidemiology and transmission patterns of concomitant genital chlamydial and gonococcal infections

Although *Chlamydia trachomatis* is a major cause of post-gonococcal urethritis,[1,2] little is known about the epidemiology and associated clinical features of concurrent gonococcal and chlamydial infections. From previous studies, the prevalence of concomitant *Chlamydia trachomatis* among patients with uncomplicated gonorrhoea ranges from 14–42%.[1-5]

We reviewed case notes of patients diagnosed with concurrent *N gonorrhoeae* and *C trachomatis* infections in the genitourinary medicine (GUM) clinic at North Staffordshire Hospital in 1993 and 1994. The aim of the study was to describe the frequency, epidemiology and transmission patterns of concurrent gonococcal and chlamydial infections.

Diagnosis of gonorrhoea was based on positive culture (modified New York City medium). Chlamydia was diagnosed on the basis of two non-culture tests, Enzyme Immunoassay (EIA) (Syva Microtrak, UK) and Direct Immunofluorescence (DFA) (Syva

Microtrak, UK), used routinely in the hospital laboratory. Using the expanded "gold standard" suggested by Thejls *et al*[6] we considered a patient infected with *C trachomatis* if they tested positive on the two tests. There were no discordant EIA and DFA test results.

Thirty-one patient-records (13 men and 18 women) were available for analysis. Table 1 summarises the demographic, clinical and laboratory details. We also identified case-notes for 21 patients who had attended the GUM clinic as known sexual contacts of individuals with concomitant gonococcal and chlamydial infections. Table 2 summarises the clinical and laboratory findings in the 21 sexual contacts.

Of the 92 cases of gonorrhoea diagnosed in our clinic in 1993/94, 31 (33·7%) had concomitant chlamydial infection, and this is within the range reported by others.[1–5] The mean age of men and women correlates with the reported age for gonorrhoea in the west Midlands.[7] Most of the infections (84%) were acquired locally. It is reassuring that 94% attended the scheduled two tests of cure and none of the patients defaulted without treatment. Most of the men (92%) were symptomatic, compared with 39% of the women. This finding seems to emphasise the importance of assiduous contact tracing in the control of STDs in women. All the men had post-gonococcal urethritis (PGU), on microscopy, which responded to doxycycline.

Sexual contacts of 23 (74%) patients were traced. Twenty-one sexual contacts were available for analysis as shown in table 2.

Table 1 Demographic, clinical and laboratory data

Feature	*Male*	*Female*	*Total*
Number	13	18	31
Age in years (mean)	24	20	22
Number unemployed (%)	2 (15·4)	7 (38·8)	9 (29)
Number symptomatic (%)	12 (92·3)	7 (38·8)	19 (61·3)
Antibiotics in last 3 months	0	2	2
Condom used in last 3 months	1	1	2
Test of cure done	13 (100)	16 (88·8)	29 (92·5)
Source of infection (%):			
Local	11 (84·6)	15 (83·3)	26 (83·9)
UK	1	2	3
Europe	1	0	1
Africa	0	1	1
Partners in last 3 months (mean)	1·5	1·6	
Last coitus (mean days)	6·4	12·9	
Past STD	2	2	4
Mode of attendance (%):			
Self referral	5	2	7 (22·6)
GP referral	6	5	11 (35·5)
Contact slip	2	11	13 (41·9)
Contacts traced (%)	9 (69·2)	14 (77·7)	23 (74·2)

STD = Sexually transmitted disease.

Table 2 Clinical and laboratory findings in sexual contacts

Feature	*Male*	*Female*	*Total*
Number	12	9	21
Age (mean)	23·5	20·4	
Last coitus (days)	7·5	12·6	
Condom used in past 3 months	1	1	2
Antibiotics in past 3 months	0	1	1
NG and CT positive	5	6	11 (52%)
Only NG isolated	5	0	5 (25%)
Only CT positive	1	0	1 (5%)
NG and CT negative	1	3	4 (19%)

NG = *N gonorrhoeae*
CT = *C trachomatis*

Eleven (52%) patients acquired both infections, while 4 (19%) apparently eluded both infections. Transmission rate was higher for gonorrhoea than chlamydia (76% versus 52%), and this is similar to Lycke *et al*'s findings.[8] In Lycke *et al*'s study the transmission was higher for sole gonococcal infections than dual infections with chlamydia; whereas that for chlamydia was the same for sole and dual infections.[8] Reasons for this finding are as yet unknown. Although our numbers are small, on comparison, our findings are similar to Lycke *et al*'s: prevalence of gonorrhoea was higher in male than female sexual contacts; while that for chlamydia was higher in female than male sexual contacts. Lycke *et al*'s and our findings on the transmission of *N gonorrhoeae* seem to contradict studies[5] which conform to the general view that efficiency of transmission of STDs is greater male-to-female than vice versa.

Whether co-existence of *N gonorrhoeae* and *C trachomatis* alters the biological behaviour of the microorganisms, and if so in what way, requires further study.

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Infertility due to *Chlamydia trachomatis* infection: what is the appropriate site for obtaining samples?

Chlamydia trachomatis infection of the upper genital tract often leads to "silent" infertility, through producing pelvic inflammatory disease (PID). After an episode of salpingitis, the post-infection infertility rate varies from 11 to 25%[1] however, there is little information on the aetiology, pathophysiology or the magnitude of the problem of asymptomatic tubal